

Vitamin D insufficiency in a population of healthy western Canadians

Diana Rucker,^{*,†} Jane A. Allan,[†] Gordon H. Fick,[‡]
David A. Hanley[†]

Abstract

Background: People with low levels of vitamin D and its metabolites are at increased risk for osteoporotic fractures. We wished to ascertain levels of vitamin D in a representative sample of adult western Canadians, to help assess the level of risk. We evaluated the prevalence of vitamin D insufficiency, defined as 25-hydroxyvitamin D [25(OH)D] less than 40 nmol/L, and seasonal variations in 25(OH)D, parathyroid hormone and related biochemical indices in a community-dwelling population of healthy Canadians living in Calgary (latitude 51°07'N).

Methods: During calendar year 1999, we collected fasting overnight blood samples every 3 months from 60 men and 128 women (age range 27 to 89 years) who had volunteered to participate in another study. We used commercial radioimmunoassay kits to measure calciotropic hormones and other biochemical indices. Regression models for longitudinal data were used to assess the effect of season and other potential predictors on individual parameters.

Results: For a total of 64 participants (34%), vitamin D insufficiency, defined as 25(OH)D less than 40 nmol/L, was recorded at least once out of the 4 sampling times. After adjustment for age, body mass index and holiday travel, we observed the anticipated rise in serum 25(OH)D from a mean of 57.3 (standard deviation [SD] 21.3) nmol/L in the winter to 62.9 (SD 28.8) nmol/L in spring ($p = 0.001$) and 71.6 (SD 23.6) nmol/L in summer ($p < 0.001$), with a subsequent decline to 52.9 (SD 17.2) nmol/L in the fall ($p = 0.008$). The anticipated inverse relation between 25(OH)D and parathyroid hormone was not consistently observed: after adjustment for age, sex, body mass index and serum calcium, serum levels of parathyroid hormone did decrease significantly, from 39.5 (SD 18.8) ng/L in winter to 36.3 (SD 17.8) ng/L in summer ($p = 0.001$), but they continued to decline to 34.5 (SD 17.3) ng/L in the fall ($p < 0.001$). There was no association between 25(OH)D and parathyroid hormone ($p = 0.21$).

Interpretation: We documented a high prevalence of vitamin D insufficiency, which warrants consideration of dietary vitamin D supplementation.

Vitamin D deficiency has long been recognized as a cause of rickets in children and osteomalacia in adults. More recent is the awareness of a preclinical phase of vitamin D deficiency, known as vitamin D insufficiency, which increases the risk of osteoporotic fractures.¹⁻³ Low levels of vitamin D metabolites are associated with malabsorption of calcium, which results in bone loss.^{4,5} Vitamin D can be obtained through the diet or it is synthesized in the skin after exposure to the sun. However, because few foods provide a natural source of vitamin D⁶ and because fortification of foods with vitamin D is often unreliable,^{7,8} skin synthesis is thought to constitute the major source. People living in countries at higher latitudes are more prone to seasonal vitamin D insufficiency because wintertime sunlight does not promote conversion of the vitamin D precursor in the skin.⁹ Levels of vitamin D and its main circulating metabolite, 25-hydroxy vitamin D [25(OH)D], are under the predominant influence of solar ultraviolet B radiation

Research

Recherche

From the Departments of
*Medical Science, †Medicine
and ‡Community Health
Sciences, University of
Calgary, Calgary, Alta.

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(wavelength 290 to 315 nm). Webb and colleagues⁹ have shown that in Boston (latitude 42°N), sun irradiation is inadequate to generate previtamin D *in vitro* from November through February; in Edmonton (latitude 53°30'N), this period extends from October through March.

The vitamin D hormone system and parathyroid hormone are the principal regulators of serum concentrations of calcium and therefore influence skeletal calcium reserves. Because of the health risks associated with low levels of vitamin D, our objective was to determine patterns of seasonal variation in 25(OH)D, as well as the prevalence of vitamin D insufficiency, defined as 25(OH)D less than 40 nmol/L,¹⁰ in a population of healthy men and women living in western Canada. Although 1,25-dihydroxy vitamin D [1,25-(OH)₂D] is the most potent form of vitamin D, 25(OH)D is the main circulating metabolite of vitamin D and is considered the correct functional indicator of vitamin D stores in humans.^{11,12} We also examined associations between 25(OH)D and serum concentrations of 1,25-(OH)₂D, parathyroid hormone and other related biochemical markers.

Methods

Participants for this study were recruited from participants in the Canadian Multicentre Osteoporosis Study (CaMOS). Details of the design and methods for CaMOS have been described elsewhere.¹³ In brief, CaMOS involves a population-based, randomly selected group of approximately 9000 Canadians 25 years of age and older, distributed across 9 study centres. The number of participants at each centre reflects relative population size at that location, and age and sex distribution (approximately 30% men) is similar for all centres. Those invited to participate in the vitamin D study were randomly selected (by a computerized random selection process) from the Calgary cohort of CaMOS ($n = 1065$) in age and sex proportions corresponding to those of the main study. Of 463 people contacted by telephone, 204 subjects, ranging from 27 to 89 years of age, agreed to participate. All participants were healthy and ambulatory and were dwelling in the community. Exclusion criteria were vitamin D deficiency [serum 25(OH)D below 25 nmol/L] on prestudy screening or daily consumption of vitamin D supplements providing more than 200 IU daily. Participants were instructed to not exceed this intake for the duration of the study. The University of Calgary Conjoint Medical Research Ethics Board approved the study, and all participants provided informed written consent.

During calendar year 1999, each participant came to the centre for testing 4 times, at 3-month intervals (in February or March, May or June, August or September, and November or December). Blood samples were collected between 7:30 am and 10:30 am after an overnight fast. Height and weight were measured with a stadiometer and balance scale respectively after participants had removed their shoes. Body mass index was calculated as weight divided by the square of height (kilograms per square metre). At each visit, participants were interviewed to determine holiday travel of at least 1 day at a latitude below 40°N and vitamin D supplementation up to 200 IU/day. Dietary calcium intake, smoking status, alcohol intake, self-reported ethnicity and menopausal status (for women) were obtained from the CaMOS

baseline questionnaires that had been administered 2 years previously. Dietary calcium intake was estimated by means of an abbreviated food frequency questionnaire administered by a skilled technician, who used food models to help participants estimate portion sizes. Serum levels of total calcium, inorganic phosphate, alkaline phosphatase and creatinine (to check for renal insufficiency) were measured with a Roche Hitachi autoanalyzer (Roche Diagnostics, Laval, Que.) on the same day. Blood samples for 25(OH)D, 1,25-(OH)₂D, parathyroid hormone and osteocalcin were frozen at -80°C and analyzed in batches within the next month. Both 25(OH)D and 1,25-(OH)₂D were measured in 2-step procedures with commercial immunoradiometric assay kits (DiaSorin Inc., Stillwater, Minn.). These assays involved a preliminary extraction and purification, followed by addition of antibody and tracer specific for the 2 metabolites of interest. The intra- and inter-assay coefficients of variation were 11.7% to 12.5% and 9.4% to 11.0% respectively for 25(OH)D and 10.5% to 13.1% and 9.2% to 10.8% respectively for 1,25-(OH)₂D. Intact serum parathyroid hormone and osteocalcin were measured by a one-step commercial immunoradiometric assay (DiaSorin Inc.). The intra- and inter-assay coefficients of variation were 2.4% to 3.6% and 3.4% to 4.9% respectively for parathyroid hormone and 4.5% to 6.3% and 7.1% to 9.5% respectively for osteocalcin.

Data are presented as means (and standard deviation [SD]) unless otherwise noted. Log transformations of body mass index, 25(OH)D, 1,25-(OH)₂D, parathyroid hormone and osteocalcin were used in the regression analysis to correct for right-skewed distributions, but the data are presented as means (and SD) for ease of interpretation. Regression models for longitudinal data¹⁴ were used to assess seasonal variations in biochemical indices and other potential predictors. Separate models were created for 25(OH)D, 1,25-(OH)₂D, parathyroid hormone, total calcium, inorganic phosphate, alkaline phosphatase and osteocalcin. Interaction variables were created to assess possible effect modification between age and BMI, as well as all other covariates that changed with season (e.g., holiday travel or other biochemical markers). Nonsignificant predictors were removed from the final model. A p value less than 0.05 (2-sided test) was considered statistically significant.

Results

Of the 204 subjects recruited, 188 completed the study. Three women were excluded before the study began because their serum level of 25(OH)D was less than 25 nmol/L. An additional 13 participants dropped out during the study for various reasons: 3 women began taking vitamin D supplements greater than 200 IU/day, 4 women and 1 man could not fulfill the time commitment, 3 men and 1 woman did not show up on test days, and 1 woman died.

There were no significant differences between men and women in terms of age or BMI ($p = 0.52$) (Table 1). Of the 188 subjects who completed the study, 3 had identified themselves on entry to CaMOS as members of an ethnic minority; the other 185 subjects did not identify themselves as members of an ethnic minority.

Seasonal changes in calciotropic hormones and related biochemical markers are presented in Table 2; the changes in 25(OH)D, 1,25-(OH)₂D and parathyroid hormone are

presented graphically in Fig. 1. Other significant predictors in the seasonal regression model for each biochemical marker are summarized in Table 3.

As anticipated, there was a significant rise in serum 25(OH)D during spring (May or June) and summer (August or September) relative to winter values (February or March) (Table 2). In the fall (November or December), 25(OH)D levels declined to significantly lower levels than those observed during the winter months (Table 2). There

were also several other independent predictors of 25(OH)D: both increasing age and increasing BMI were associated with significant decreases in 25(OH)D, whereas travel to lower latitudes (below 42°N) for at least 1 day was associated with significant increases in 25(OH)D (Table 3). Because individual travel to lower latitudes was not consistent for each season, we tested for, but did not find, any interactions between travel and season. This apparent lack of significant interactions may be influenced in part by the

Table 1. Baseline characteristics of 188 healthy residents of Calgary for whom vitamin D and related biochemical indices were measured seasonally for 1 year

Characteristic	Sex; mean (and SD)*	
	Men n = 60	Women n = 128
Age, yr	63.8 (11.9)	64.3 (12.7)
Weight, kg	82.1 (14.3)	71.1 (16.5)
Height, cm	175.3 (7.2)	161.6 (6.5)
Body mass index, kg/m ²	26.7 (3.9)	27.3 (6.5)
Dietary calcium intake, mg/day†	807 (544)	882 (602)
No. (and %) with history of smoking > 6 mot	32 (53)	40 (31)
No. (and %) of current smokers > 6 mot	4 (7)	13 (10)
No. (and %) consuming > 7 alcoholic beverages/ wkt	9 (15)	17 (13)
No. (and %) menopausal (surgical or natural)†	NA	102 (80)

Note: SD = standard deviation, NA = not applicable.

*Except where indicated otherwise.

†Data obtained from baseline questionnaires for the Canadian Multicentre Osteoporosis Study, completed 2 years before the current study.

Table 2. Seasonal changes in calcitrophic hormones and related biochemical markers

Hormone or biochemical marker	Season; mean concentration (and SD)*				Normal range
	Winter (baseline)	Spring	Summer	Fall	
Serum 25(OH)D, nmol/L†‡	57.3 (21.3)	62.9 (28.8)	71.6 (23.6)	52.9 (17.2)	40–130
		(p = 0.001)	(p < 0.001)	(p = 0.008)	
Serum 1,25-(OH) ₂ D, pmol/L†§	168.1 (91.5)	148.9 (83.4)	142.0 (68.9)	128.9 (50.8)	45–145
		(p < 0.001)	(p = 0.001)	(p < 0.001)	
Serum PTH, ng/L†¶	39.5 (18.8)	39.3 (18.7)	36.3 (17.8)	34.5 (17.3)	13–54
		(p = 0.81)	(p = 0.001)	(p < 0.001)	
Serum calcium, mmol/L**	2.3 (0.09)	2.29 (0.11)	2.28 (0.09)	2.27 (0.09)	2.10–2.55
		(p = 0.08)	(p = 0.004)	(p = 0.001)	
Serum inorganic phosphate, mmol/L††	1.05 (0.15)	1.03 (0.14)	1.09 (0.14)	1.10 (0.15)	0.8–1.6
		(p = 0.29)	(p < 0.001)	(p < 0.001)	
Serum alkaline phosphatase, U/L†‡	80 (24)	80 (24)	83 (23)	86 (24)	39–117
		(p = 0.73)	(p < 0.001)	(p < 0.001)	
Serum osteocalcin, nmol/L†§§	1.08 (0.54)	1.09 (0.54)	1.12 (0.56)	1.18 (0.59)	0.46–2.07
		(p = 0.46)	(p = 0.008)	(p < 0.001)	

Note: 25(OH)D = 25-hydroxy vitamin D; 1,25-(OH)₂D = 1,25-dihydroxy vitamin D; PTH = parathyroid hormone.

*p values relate to within-subject comparisons of seasonal measurements with the baseline (winter) measurement.

†Log transformations were used in the regression analysis to correct right-skewed data.

‡p values adjusted for age, body mass index (BMI) and holiday travel to lower latitudes.

§p values adjusted for age, BMI, serum PTH, serum 25(OH)D and serum inorganic phosphate.

¶p values adjusted for age, sex, BMI and serum calcium.

**p values adjusted for age and serum PTH.

††p values adjusted for sex, serum PTH and serum calcium.

‡‡p values adjusted for age, BMI and serum calcium.

§§p values adjusted for serum calcium.

relatively low number of people who travelled to lower latitudes: 22 participants (12%) during the winter, 16 (8%) during the spring, 1 (1%) in the summer and 12 (6%) in the fall. Also worth noting is that sex ($p = 0.61$) and low-level vitamin D supplementation ($p = 0.28$) were not significant predictors of 25(OH)D.

1,25-(OH)₂D decreased significantly from above the normal range in winter to close to or within the normal range in spring, summer and fall (Table 2). In addition to being associated with season, lower 1,25-(OH)₂D levels were associated with increasing age and BMI (Table 3). Finally, the complex regulation of 1,25-(OH)₂D was supported by the

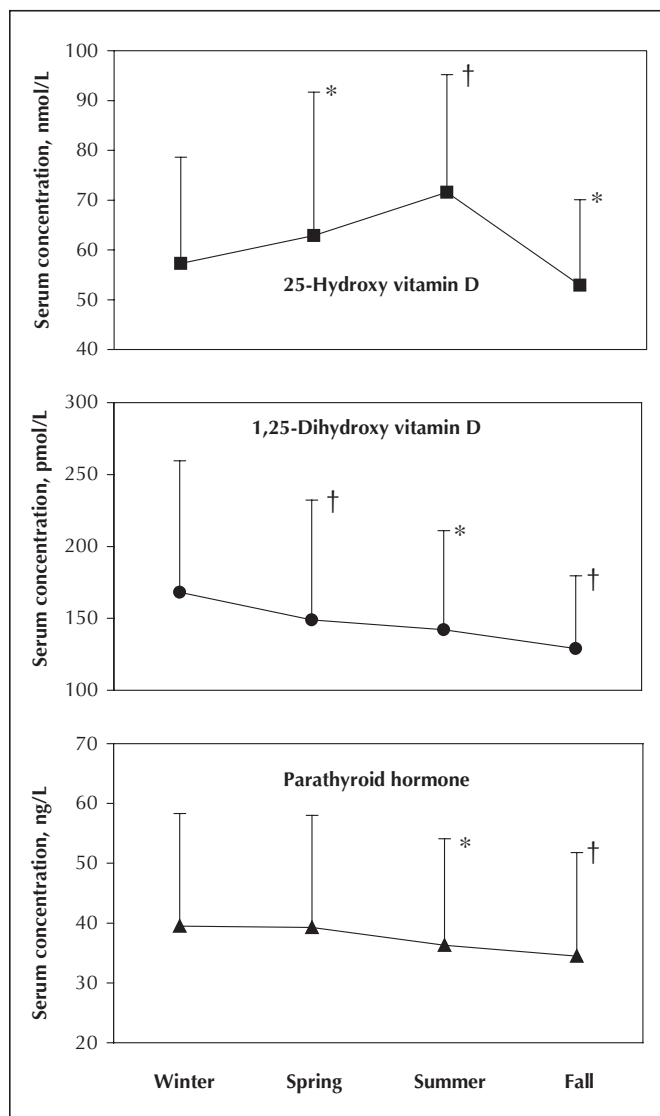


Fig. 1: Mean seasonal values for 25-hydroxy vitamin D, parathyroid hormone and 1,25-dihydroxy vitamin D. The vertical lines represent standard deviation of the means. The p values refer to within-subject comparisons of seasonal measurements with the baseline (winter) measurement (* $p < 0.01$; † $p < 0.001$) as determined by longitudinal regression models adjusted for other significant predictors (see Table 2).

biochemical parameters significant in the model: both 25(OH)D and parathyroid hormone were positively associated with 1,25-(OH)₂D, whereas inorganic phosphate was negatively associated with this metabolite (Table 3).

In contrast to the trends for 25(OH)D, serum levels of parathyroid hormone decreased significantly in the summer and in the fall, so that the anticipated inverse relation between 25(OH)D and parathyroid hormone was not consistently observed (Table 2). In addition to season, age, sex, BMI and serum calcium were significant independent predictors of parathyroid hormone levels (Table 3). 25(OH)D was not a significant predictor of parathyroid hormone ($p = 0.21$).

In conjunction with the declines in parathyroid hormone levels in the summer and fall, serum inorganic phosphate increased during the same seasons (Table 2). Serum levels of alkaline phosphatase and osteocalcin also increased in these 2 seasons (Table 2). The latter were unusual and unexpected findings, because these markers are expected to decrease with decreases in parathyroid hormone. Although the changes in inorganic phosphate, alkaline phosphatase and osteocalcin during the summer and fall were statistically significant, the mean values were all within the normal reference ranges (Table 2).

The prevalence of vitamin D insufficiency according to several different thresholds (less than 40, less than 50 and less than 80 nmol/L) is shown in Fig. 2. The prevalence of vitamin D insufficiency was generally lowest in the summer and highest in the fall. A total of 64 (34%) of the participants had 25(OH)D levels less than 40 nmol/L for at least one point during the year, 115 (61%) had levels less than 50 nmol/L for at least one point, and 182 (97%) had levels less than 80 nmol/L for at least one point.

Interpretation

We documented seasonal variations in levels of 25(OH)D and a high prevalence of vitamin D insufficiency during fall and winter in an ambulatory population of healthy western Canadians situated at 51°07'N. Although seasonal variations in serum 25(OH)D in healthy populations have been documented for several European countries and the United States,¹⁵ studies of the seasonal effects on vitamin D status in Canadians have been limited to elderly people living in institutions,¹⁶⁻¹⁸ low-income elderly people,¹⁹ and young women.²⁰ In accord with the in vitro evidence of Webb and colleagues⁹ that, at a latitude of 53°N, the skin does not produce any precursors for vitamin D₃ (the form generated by exposure to ultraviolet radiation) from October through March, we found that levels of 25(OH)D in late fall (virtually all measurements were obtained during November) were similar to those obtained in winter. However, we were surprised that the serum levels of 25(OH)D in the fall were significantly lower than the winter levels, given the relatively small absolute difference (4.4 nmol/L) between the 2 seasons (Table 2).

Seasonal changes in serum 25(OH)D levels may reflect synthesis of both vitamin D₃ and 25(OH)D. Gascon-Barré and associates²¹ demonstrated an increase in expression of the gene for vitamin D₃-25 hydroxylase (*CYP27A*) during spring and summer, with corresponding increases in serum 25(OH)D. These results were corroborated by Lehmann and collaborators,²² who demonstrated that both vitamin D₃ and exposure to ultraviolet B radiation led to increases in expression of D₃-25 hydroxylase in keratinocytes.

This study also confirmed the importance of age as a major determinant of vitamin D status. Regardless of season, increasing age was associated with lower levels of 25(OH)D and 1,25-(OH)₂D. The cause of the age-related decline in 1,25-(OH)₂D is multifactorial. Several theories, including a decrease in renal 1 α -hydroxylase activity with age,²³ compounded by decreased renal function,⁴ and decreased availability of 25(OH)D as a substrate,²⁴ have all been implicated in the age-related decline of 1,25-(OH)₂D. Declining levels of 25(OH)D with age have been attributed to impaired vitamin D absorption from the intestine,²⁵ as well as a decline in the concentration of the vitamin D precursors that are normally stored in the skin.²⁶

After adjusting for season, age and travel to lower latitudes, we found that BMI was inversely related to serum 25(OH)D. Such a relation has also been observed in postmenopausal women,²⁷ elderly people,^{1,28} and younger obese subjects.²⁹ Need and associates²⁷ suggested that the inverse relation between 25(OH)D and BMI is due a larger body pool of vitamin D and 25(OH)D or slower

saturation and mobilization of these compounds from adipose tissue (or both).

Serum 25(OH)D is our best available laboratory aid for diagnosing frank vitamin D deficiency, which causes rickets

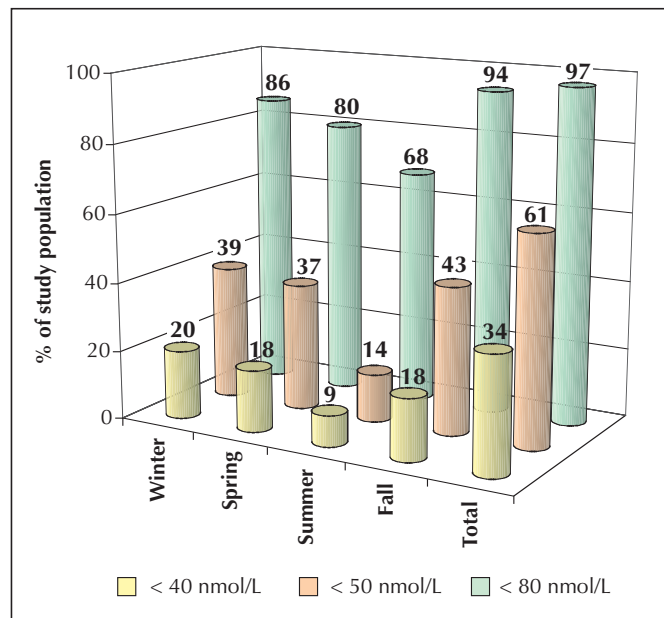


Fig. 2: Prevalence of vitamin D insufficiency, defined in terms of serum level of 25-hydroxy vitamin D. The data for “total” represent the percentages of subjects with vitamin D insufficiency at least once during the year.

Table 3: Additional significant predictors of calciotropic hormones and biochemical markers in seasonal regression model

Hormone or biochemical marker	Significant predictor	p value
Serum 25(OH)D (nmol/L)	Age, yr	0.007*
	BMI, kg/m ²	< 0.001*
	Travel to lower latitude (< 42°N for ≥ 1 day)	0.002
Serum 1,25-OH ₂ D (pmol/L)	Age, yr	< 0.001*
	BMI, kg/m ²	< 0.001*
	Serum PTH, ng/L	< 0.001
	Serum 25(OH)D	0.002
	Serum inorganic phosphate	0.001*
Serum PTH (ng/L)	Age, yr	< 0.001
	Sex (male compared with female)	0.022
	BMI, kg/m ²	0.006
	Serum calcium	< 0.001*
Serum calcium (mmol/L)	Age, yr	0.001
	Serum PTH, ng/L	< 0.001*
Serum inorganic phosphate (mmol/L)	Sex (male compared with female)	< 0.001
	Serum PTH, ng/L	0.005*
	Serum calcium	< 0.001
Serum alkaline phosphatase (U/L)	Age, yr	< 0.001
	BMI, kg/m ²	0.007
	Serum calcium, mmol/L	< 0.001
Serum osteocalcin (nmol/L)	Serum calcium, mmol/L	0.001

*Inverse relation.

in children and osteomalacia in adults. Vitamin D deficiency is typically associated with 25(OH)D levels below 25 nmol/L.³⁰ However, identification of inadequate levels of vitamin D for optimal bone health — vitamin D “insufficiency” — is likely of greater clinical importance. The most commonly used definition of vitamin D insufficiency is the lower limit of the normal range for the 25(OH)D assay (40 nmol/L).¹⁰ However, recent investigations have suggested that the threshold for vitamin D insufficiency should be the 25(OH)D level below which parathyroid hormone secretion begins to rise. The authors of these studies have proposed that the cutoff value for vitamin D insufficiency may be as high as 80 nmol/L.^{31–33} In support of a higher threshold, it has been noted that serum 25(OH)D is lower in people who have experienced fractures than in controls.^{34,35} Center and colleagues³⁶ showed that subjects with a 25(OH)D level less than 68 nmol/L had a 4-fold greater risk of major fracture over an 8-year period. In patients with osteoporosis this risk was increased 19-fold.

On the basis of the most conservative definition of insufficiency (less than 40 nmol/L), 34% of our subjects had vitamin D insufficiency at at least one point during the year (Fig. 2). If the proposed 80 nmol/L threshold is used, virtually all participants (97%) would be assessed as having had vitamin D insufficiency at least once during the year. Raising the threshold by only 10 nmol/L, to 50 nmol/L, nearly doubles the proportion of our study population who would be classified as having vitamin D insufficiency (to 61%).

Despite the high prevalence of vitamin D insufficiency in this population and appropriate seasonal changes, we found no relation between parathyroid hormone and serum 25(OH)D. Only a small proportion (10% to 17%) of participants had parathyroid hormone levels above the normal range of the DiaSorin assay (greater than 54 pg/mL) at any point. Similar to other studies,^{37,38} we observed an increase in parathyroid hormone with age, and women had higher levels of this hormone than men. In addition, parathyroid hormone increased with increasing BMI. The seasonal declines in parathyroid hormone, which lagged behind seasonal increases in 25(OH)D by one season, may be the re-

sult of previously established vitamin D stores in body fat.³⁹ Another possible reason for the lack of an association between parathyroid hormone and 25(OH)D is that other factors, particularly calcium consumption, may have served to keep serum parathyroid hormone levels low, even during times of 25(OH)D insufficiency. Both Riggs and collaborators⁴⁰ and Storm and associates⁴¹ reported wintertime suppression of parathyroid hormone with calcium supplementation (without dietary vitamin D supplementation) in postmenopausal women. In both of these studies, mean daily calcium intake was well over 1500 mg in each of the intervention groups. Storm and associates⁴¹ also reported a significant inverse relation between parathyroid hormone and 25(OH)D only when calcium consumption was less than 800 mg/day, which suggests a threshold dose of calcium for suppression of parathyroid hormone. An important limitation of our study is that we did not assess participants' dietary calcium during the study. Although the subjects had all reported relatively low calcium intake on entry into CaMOS 2 years earlier (Table 1), they probably increased their calcium intake after entering the study, as a result of their participation in an “osteoporosis study.”

This study has several other limitations that must be considered when generalizing the results to other Canadian populations. The study sample was predominately white (except for 3 individuals), which does not accurately reflect more diverse ethnic communities such as those in Vancouver and Toronto. However, given that people with greater skin pigmentation have less circulating vitamin D₃ than those with lighter skin tone,^{42,43} it is likely that a larger proportion of dark-skinned participants would have reduced the mean serum 25(OH)D levels throughout the year. Other factors unique to the Calgary population might have led to an underestimation of the seasonal variations of 25(OH)D in the Canadian population as a whole. Calgary receives more hours of sunshine per year than any other Canadian city, particularly in the fall and winter (Table 4).⁴⁴ Although sunshine hours do not accurately indicate how much time people spend in the sun, several studies have reported a direct relation between meteorologically measured

Table 4: Normal duration* of sunshine† in the fall and winter in various Canadian cities⁴⁴

City	Latitude, N	Elevation, m	Month; total duration of sunshine, h							Total
			Oct	Nov	Dec	Jan	Feb	Mar		
Whitehorse	60°43'	703	90	45	25	46	98	161	465	
Edmonton	53°30'	668	165	101	78	95	117	170	726	
Calgary	51°07'	1077	179	125	102	114	137	174	831	
Vancouver	49°15'	86	122	67	53	55	82	130	509	
Victoria	48°39'	20	139	76	57	64	90	143	569	
St. John's	47°37'	114	105	72	60	75	88	107	507	
Quebec City	46°48'	102	122	79	82	102	121	150	656	
Toronto	43°40'	112	145	82	73	96	113	150	659	

*Normal duration of sunshine, in hours, is the monthly average over a 30-year period (about 1960 to 1990).

†Sunshine observations are made with the Stokes-Campbell sunshine recorder, which measures only “bright” sunshine (but not “visible” sunshine). For example, sunshine immediately after sunrise and just before sunset would not be bright enough to be registered with this recorder.

sunshine hours and levels of 25(OH)D.^{27,31} In addition, at an elevation of 1077 m, Calgary residents are situated at a considerably higher altitude than most other Canadians (Table 4). Rigel and colleagues⁴⁵ have demonstrated increases in ultraviolet B radiation of approximately 8% to 10% with every 300 m increase in elevation. Given that participants in this study lived in a region with more sunshine hours and higher elevation than most other Canadian locations, it is likely that the seasonal decreases observed in late fall and winter underestimate those of other Canadian populations. These factors also suggest that the observed prevalence of seasonal vitamin D insufficiency in a Calgary population may be a conservative estimate of vitamin D insufficiency among other Canadians.

In conclusion, we documented marked seasonal effects on vitamin D levels in a community-dwelling sample of Canadians. These results support recommendations for clinicians recently outlined by Heaney⁴⁶ regarding assessment of vitamin D metabolism. According to Heaney, the reference values provided by manufacturers of 25(OH)D measurement kits should be revised. Experts suggest that optimal levels of 25(OH)D should be somewhere above 80 nmol/L (32 ng/mL).^{31,46} Furthermore, we do not recommend using elevated serum levels of parathyroid hormone as a surrogate marker for vitamin D insufficiency, because parathyroid hormone is modulated by several other factors, particularly calcium intake. However, despite these expert suggestions, routine measurement of 25(OH)D is probably not justified.⁴⁶ Given that almost every person in our sample had serum 25(OH)D levels below 80 nmol/L at one point in the year, and that more than one-third of subjects had levels below the most conservative definition of vitamin D insufficiency, our findings support a recommendation for more aggressive vitamin D supplementation, particularly for elderly people and especially during the fall and winter months. The current dietary recommendation for vitamin D in Canada and the United States is 200 IU/day for adults (600 IU/day for those over the age of 70),⁴⁷ although Vieth⁴⁸ has argued that the adult requirement should be at least 800 IU/day. Our study suggests a need to review Canada's recommendation for vitamin D nutrition.

Competing interests: None declared.

Contributors: Ms. Rucker assisted with data collection and performed the data entry, cleanup and analysis, as well as contributing to the initial writing of the manuscript. Jane Allan was responsible for coordinating the project, and Dr. Fick contributed to the statistical methods and the interpretation of the data. Dr. Hanley was the principal investigator responsible for project conception, study design, grant application and supervision of the writing of the final manuscript. All authors contributed to reviewing and making revisions to the manuscript.

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Correspondence to: Dr. David A. Hanley, 3330 Hospital Dr. NW, Calgary AB T2N 4N1; fax 403 270-0979; dahanley@ucalgary.ca

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